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"It is as hard to live in this world with your lover out of order as it is to love with your liver out of order."

—*G. F. Butler.*

DIAGNOSIS AND TREATMENT

OF

DISEASES
OF THE LIVER

HENRY IRVING BERGER, M. D.

PUBLISHED BY
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1913

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Introduction

A BRIEF tabulation of the functions of the liver will undoubtedly impress the reader with the great physiologic importance of the liver (the largest organ of the body), and it will also show the enormous working field for physiologists, pathologists, and clinicians; namely, the recognition of the functional derangements of the liver, as well as the diseases resulting directly from the alteration in structure of the hepatic cells, and, lastly, many gastro-intestinal diseases.

The functions ascribed to the liver are:

1. The formation of bile. Bile is principally concerned in the digestion of fat.

2. Glycogenic function. The conversion of carbohydrates into glycogen and the reconversion of glycogen into sugar.

3. The conversion of amino bodies formed during the process of digestion into urea.

4. Antitoxic or protective function, which is commonly referred to as the "detoxifying action of the liver."

The proper functions of the liver are dependent upon the integrity of the liver cells. When for any reason the hepatic cells become morphologically altered we must expect serious interference in the health of the individual directly in proportion to the degree of damage to the cells in question. Either one or more of the functions may be affected.

While there are many primary diseases of the liver we have a goodly number resulting from changes elsewhere in the body. For this reason the proper understanding of the hepatic diseases requires that one familiarize himself with the following: (a) diseases

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of the heart which may cause passive congestion of the liver, (b) pulmonary diseases and diseases of other intrathoracic structures which may also cause hepatic congestion, (c) and last, but not least, gastro-intestinal disorders. The latter are extremely important owing to the intimate association of the liver with the intestines in the processes of digestion and the analogous symptoms appearing in the diseases of these structures. Furthermore, anatomy teaches us that the tributaries of the portal vein arise principally from stomach, spleen, and the intestines, hence, it is very easy to see that suppurative processes in the pathway of the portal vein may burrow therein and ultimately cause abscesses of the liver. Figuratively speaking of the circulation of the liver we might liken the liver to a wedge placed between the heart and lungs on one side and the gastro-intestinal tract on the other.

This booklet will be chiefly devoted to the diagnosis of the various hepatic disorders. The text is simple and yet very comprehensive. The value of urinalysis and the X-ray in the diagnosis of diseases of the liver is fully discussed. Great emphasis has also been given to the differential diagnosis of such conditions as jaundice, cirrhosis, enlargements of the liver, etc.

It is hoped that the student, as well as the practitioner, will find this booklet a valuable help and guide in the proper understanding and interpretation of the diseases of the liver.

HENRY IRVING BERGER,
Indianapolis, Ind.

Anatomical Facts Necessary in the Diagnosis of Hepatic Diseases

The liver is the largest organ in the body and occupies three regions: the right hypochondrium, the epigastrium, and the left hypochondrium.

The liver moves up and down with respiration (the ascent and descent of the diaphragm), but the liver has no motion of its own. However, in certain diseases of the heart, the liver may show pulsations. An exceedingly movable liver occurs as a part of a general splanchnoptosis in neurasthenic individuals. The movability of the liver is lessened in capsular cirrhosis and in certain diseases of the right pleura.

The liver changes position according to the posture assumed. In the recumbent posture, the liver retreats towards the diaphragm. On standing, it goes downward.

The liver (even when normal) may be pushed up or down by diseases of the surrounding structures. If pushed downward the liver may be thought to be enlarged. For convenience we may term such enlargements of the liver as "spurious enlargements." Some diseases of the thoracic cavity, such as a right pleurisy with effusion, a subphrenic abscess, and emphysema, will depress the liver. On the other hand, abdominal enlargements which encroach upon the thoracic cavity, no matter if the enlargement be due to gas, fluid or tumor, will push the liver upward.

The liver increases slightly in size during the process of digestion. It is explained by an increased flow of blood through this organ, and it is only temporary.

In women we find a liver spoken of as a "Corset Liver," which is due to tight lacing. It causes a transverse groove to appear on the right lobe. It predisposes to hepatic congestion and to gallstones. In a majority of these cases the liver may be felt below the costal margin.

The X-ray is a valuable instrument to determine changes in position, alteration in size, shape and consistency of the liver, inasmuch as the liver substance is opaque to the X-ray and that the liver is surrounded by more or less transparent organs. A clear and sharp profile of the liver can be seen on the screen and can be readily distinguished from the light pulmonary zone. The upper limit of the liver can be better elicited by means of the X-ray than by percussion. In some cases the character of the surface and inequalities of its outlines are the important factors in arriving at the proper diagnosis. To obtain a better view of the lower surface and lower border of the liver, the stomach should be (if necessary) distended with gas.

Owing to the structure of the hepatic veins, the veins being contained within rigid canals and devoid of valves, ruptures and severe lacerations of the liver are attended by severe hemorrhages which may result in death.

Pain over the right shoulder is significant of hepatic disease and can be explained by the anatomical course and distribution of the right phrenic nerve.

Enlargement of the Liver

Being familiar with the surface markings of the liver we should then be able to detect any increase in size of this organ by combined palpation and percussion and the use of the X-ray in doubtful cases. It must, however, be remembered that the liver may enlarge upward (as is the case in solitary abscess of the right lobe) as well as downward.

Before one can be positively certain that the liver itself is enlarged, the following must be excluded:

(A) **In cases of upward enlargements:**

1. Consolidation of lower lobe of right lung.
2. Right pleural effusions.
3. Subphrenic abscess.

(The usual cause of upward enlargement is a solitary abscess in the right lobe.)

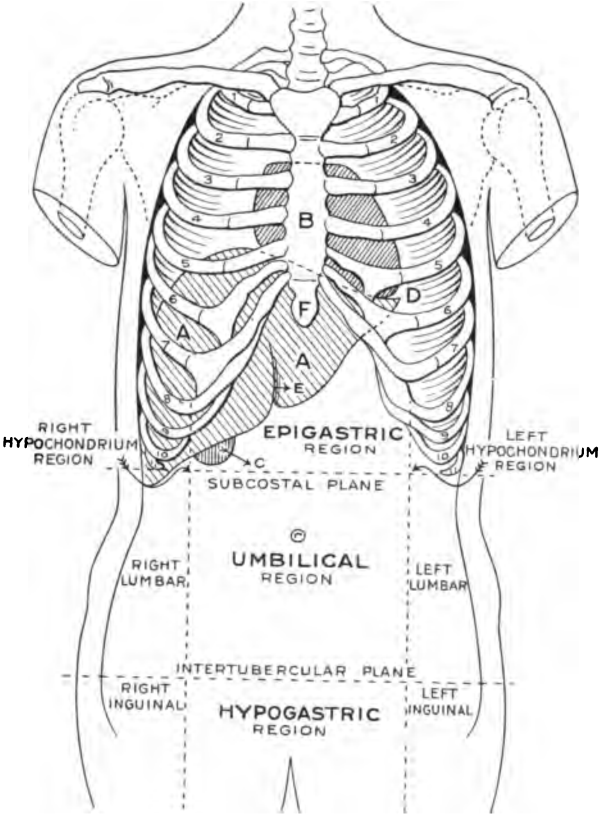
(B) **In cases of downward enlargements:**

1. Impacted feces in the transverse colon.
2. Cancer or tuberculosis of the omentum.
3. Movable liver.

The enlargements of the liver have been arbitrarily classified as (a) **local** and (b) **distant.**

Local causes:

1. Cancer of the liver.
2. Syphilis of the liver.
3. Atrophic cirrhosis (in the early stage).
4. Hypertrophic cirrhosis.
5. Solitary abscess of the liver.
6. Catarrhal jaundice.
7. Passive congestion of the liver.



Topographical Chart of the Liver, Gall-Bladder and Heart, and of the Regions of the Abdomen

A—A′ The liver.
B The cardiac Region.
C The gall-bladder at the level of the ninth costal cartilage.
D The apex of the heart.
E The falciform ligament dividing the liver into right and left lobes.
F The ensiform cartilage.

Differential Diagnosis of Enlargements of the Liver

	Character of the Enlargement	Spleen	Jaundice	Ascites	Duration	Other Signs
CANCER........	Irregular nodules on surface; some show central depression, progressive and painful enlargement.	Rarely enlarged.	Frequent.	Rare.	Never more than 1½ yrs.	Cachexia, anemia, wasting, vomiting.
SYPHILIS.......	Irregular nodules, but not umbilicated, felt on surface; liver very painful.	More often enlarged than in cancer.	Slight.	Present.	Runs a long course; will yield to KI.	Gumma elsewhere.
HYPERTROPHIC CIRRHOSIS........	Smooth, progressive enlargement; liver tender on pressure; recurrent attacks of hepatic pain.	Rarely enlarged.	Very intense.	Absent.	Several years.	
SOLITARY ABSCESS......	Enlargement upward to the right and behind. If felt below costal margin it is smooth. Dull aching in hepatic region.		Slight.			Signs of sepsis: chill, fever, sweat, etc.
CATARRHAL JAUNDICE....	Liver is smooth, enlargement slight. No pain over liver.	Absent.	Present.	Absent.	6-8 weeks.	Gastro-intestinal symptoms precede jaundice.
PASSIVE CONGESTION.	Smooth, enlarged, pulsating, tender liver, hepatic discomfort.		Slight.	Comes and goes.	Long time; depends on the heart.	Signs of valvular disease, emphysema, etc.; gastro-intestinal disturbances.
PHOSPHORUS POISONING...	Enlarged and painful.	Seldom enlarged.	Present late			Vomit, severe epigastric pains.
AMYLOID LIVER........	Progressive, uniform, very large, painless.		Absent unless complicated by gumma.	Absent unless complicated by gumma, cirrhosis, or renal disease.		Never occurs alone but as a part of a widespread lardaceous disease.
FATTY LIVER.........	Smooth, painless enlargement.		Absent.	Absent.		Those depending on the accompanying disease, as alcoholism, obesity, tuberculosis, poisoning.

SUPPURATIVE CHOLANGITIS	Both liver and gall-bladder enlarged. Liver is tender.		Present.			History of Gall-Stones. Signs of Sepsis.
OBSTRUCTION OF THE COMMON DUCT........	Only slight enlargement.	Enlarged.	Varies in intensity from time to time.			History of Biliary Colics.
SPLENO-MEDULLARY LEUKEMIA...	Smooth enlargement.	Enormous spleen.				Typical blood picture: 300,000 W.B.C. with 20% myelocytes, and R.B.C. :: W.B.C. :: 10:1 (normally it is 55:1).

Differential Diagnosis of Reductions in Size of the Liver

	Character	Spleen	Jaundice	Obstructive Symptoms	Urine	Nervous Symptoms
ATROPHIC CIRRHOSIS...	In the last stages the liver is rough, small and granular.	Enlarged.	Mild. Occurs late.	Ascites very marked. Chronic gastric catarrh, piles, hematemesis.		May occur suddenly at any time, due to absorption of toxins.
ACUTE YELLOW ATROPHY.....	Reduction most marked toward end of the disease. Some pain in hepatic area.	Markedly enlarged.	Appears early and deepens as nervous symptoms increase.	Absent, but cutaneous and internal hemorrhages may occur anywhere.	Leucin, tyrosin. Bile.	Very intense. Headache replaced by delirium, and finally by coma.
CHRONIC PERIHEPATITIS OR CAPSULAR CIRRHOSIS....	Occurs as a part of a multiple serositis.	Decreased in size.	Not common.	Ascites recurs even after tapping. The fluid is an inflammatory exudate.	Interstitial nephritis usually co-exists.	Toxic symptoms absent.

8. Phosphorous poisoning.
9. Amyloid liver.
10. Fatty liver.
11. Bile duct diseases . . .
    - **extension of gastro-duodenal catarrh.**
    - **suppurative cholangitis.**
    - **obstruction of gall ducts.**
12. Leukemia.

Distant causes: (These are usually mechanical.)

When compensation breaks in **chronic valvular disease** of the heart; when contractures and deformities are marked in **fibroid phthisis** and when **mediastinal tumors** interfere mechanically with the circulation, we find that passive congestion of the liver occurs. A liver that shows passive congestion is enlarged and has an even surface. However, somewhat later, contraction may occur, giving rise to the so-called "nutmeg" liver.

Mediastinal tumors are frequently associated with effusions into the pleural cavity. In some cases of right pleural effusion the liver will be found not only enlarged but also mechanically depressed by the weight of the liquid above, hence the enlargement is much more marked (apparently).

In **hypertrophic emphysema** the area of hyper-resonance will encroach upon the upper confines of the liver owing to the increase in the bulk of the lung, and at the same time the liver will be thereby depressed. Should the right heart fail to compensate passive congestion ensues and the liver enlarges still further.

In **pericarditis with effusion** the left lobe of the liver can be felt depressed in the epigastrium, while the apex of the heart is displaced upward and to the left fourth intercostal space. In long standing cases the blood dams back, the liver becomes congested and enlarged, ascites and edema ensues.

In **right side empyæma** and **pleurisy with effusion** the liver is displaced downward. Percussion over the fluid region gives a flat note. This flatness must not be confused with liver dulness, because one might then be led to suspect an upward enlargement of the liver. If the level of flatness changes with the change in position of the patient; if we can map out Ellis' "S"-shaped line of flatness; if there is absence of respiratory murmur where there is fluid and if there is an increased vocal sound, bronchial breathing and Skodaic resonance above the level of the fluid, then the case is one of pleural effusion.

Jaundice

Definition: Jaundice is not a disease but a symptom. It denotes a yellowish discoloration of the skin, mucous membranes, and of the fluids of the body. It is best noticeable on the conjunctiva and on the buccal mucosa.

For diagnostic purposes we must consider:

1. **Intensity, variation, remittency,** and **duration** of jaundice:

    The color ranges from a lemon-yellow to a brownish-yellow or saffron tint.

    If the jaundice is very pronounced the cause may be one of the following (in rotation):

    (a) malignant disease (if common duct is involved).
    (b) impacted stone in common duct.
    (c) biliary cirrhosis.

    Jaundice having persisted for a long time but remitting in intensity is suggestive of one of the following:

    (a) stone impacted in common duct.
    (b) biliary cirrhosis.

    Obstructive jaundice is usually more pronounced than toxemic jaundice.

    Epidemics of jaundice suggest Weil's disease. Jaundice lasting no more than six to eight weeks is in favor of catarrhal jaundice. Jaundice that is chronic and pronounced usually means malignancy.

2. **Mode of onset:** it may be gradual or sudden:

    If jaundice appears gradually without other special or striking concomitant symptoms we must think of a tumor pressing on the bile ducts.

    Jaundice occurring unexpectedly (though sometimes rather slowly) in comparatively young, healthy persons, and especially when gastro-intestinal disturbances occur three or four days prior to the development of jaundice, we may then diagnose it as catarrhal jaundice.

3. **Age of the patient:**

    Youth: suspect catarrhal jaundice.
    30 to 45: gall-stones.
    From 45 upward: malignant disease.

4. **Its nature:**

    (a) Obstructive (either within or without the ducts).
    (b) Toxemic, including the infectious variety.

    (The terms "hematogenous" and "hepatogenous" are terms usually employed to designate the probable cause of jaundice as being in the blood or in the liver respectively, but in some cases both the blood and the liver are at fault.)

Obstructive Jaundice.	**Toxemic Jaundice.**
Character: intense, persistent	not so marked; runs shorter course (as a rule).
Clay colored stools: present	absent.
Pulse: bradycardia	usually rapid.
Cerebral symptoms: mild	usually severe.
irritability, melancholia, headache. May become severe.	Vomit, delirium, convulsions.
Not always present	mostly always present.
Hemorrhages: into skin and mucous membranes	also present.

5. Associated symptoms and signs; including the condition of the liver and gall-bladder:

The gall-bladder is found enlarged in all cases of obstruction of the cystic and the common duct, regardless of whether the obstruction is from within or without, except when the obstruction is due to gall-stones. (This is known as Courvoisier's Law.)

The gall-bladder is not enlarged in toxemic jaundice and in biliary cirrhosis.

Jaundice associated with an enlarged liver occurs in:

(a) malignant disease	Marked	uneven and nodular enlargement.
(b) hypertrophic cirrhosis		smooth enlargement.
(c) abscess	slight.	

Jaundice associated with **fever** occurs in:

1. Gall-stone colic.
2. Cancer of the liver.
3. Suppurative angiocholitis.
4. Acute infectious cholecystitis.
5. Weil's disease.
6. Yellow fever.
7. Malaria.
8. Relapsing fever.

Differential Diagnosis of Diseases accompanied by Jaundice

Catarrhal Jaundice

***Onset:** unexpected and sometimes gradual.
Intensity: moderate.
Duration: seldom more than six weeks.
Fever: usually absent.
Liver: only slightly enlarged.
Age: young, healthy persons.
Prodromes: gastro-intestinal disturbances for 3 or 4 days.

Hypertrophic Cirrhosis of the Liver

***Onset:** early in the disease.
Intensity and progress: very intense and becomes still deeper after each attack of hepatic pain.
Duration: 4 to 6 years, therefore chronic.
Fever: usually slight, but may increase at time of pain.
Liver: very much enlarged, surface is even and smooth, recurrent attacks of hepatic pain.
Spleen: enlarged, easily palpable and very hard.
Age: young persons, as a rule.

Atrophic or Alcoholic Cirrhosis

***Onset:** occurs in the last stages of the disease.
Intensity: slight.
Duration: runs a chronic course. Duration is dependent upon the collateral circulation of the liver.
Liver: (a) early: it is enlarged;
(b) late: small, contracted, rough, and granular.
Spleen: enlarged.
Obstructive symptoms: ascites, hematemesis, piles, gastric catarrh.

*** Refers to jaundice.**

Gall Stones Obstructing Common Duct

Onset: history of acute attacks of biliary colic.

Intensity: varies from time to time.

Duration: always lasts more than one year.

Liver: normal or slightly enlarged.

Spleen: enlarged.

Gall-bladder: not distended (Courvoisier's Law).

Cancer of the Liver

** **Onset:** insidious.

Intensity: moderate, but if common duct is involved (blocked) it is then very marked.

Duration: death within 3 to 15 months.

Fever: continuous pyrexia; 100° to 102°.

Liver: progressive enlargement, nodular, irregular, painful.

Spleen: rarely enlarged.

Age: adults: 45 to 60 years.

Weil's Disease

Onset: jaundice appears on the second day.

Intensity: may be very severe.

Duration: one week.

Fever: present and accompanied by severe chill, headache, and pain in calf muscles.

Liver: usually enlarged.

Spleen: usually enlarged.

Age: young and middle age persons (butchers).

Acute Yellow Atrophy

Onset: the disease sets in suddenly.

Intensity: jaundice occurs early and deepens as the cerebral symptoms increase.

Duration: after the cerebral symptoms have reached their height, death occurs within 4 days.

Liver: becomes reduced in size as the end approaches.

Spleen: markedly enlarged.

**** Refers to both the disease and jaundice.**

Phosphorus Poisoning

Onset: while the disease sets in suddenly, jaundice appears later, after the quiescent period.

Intensity: jaundice is slight.

Liver: enlarged and painful.

Spleen: may be enlarged.

Yellow Fever

Onset: the disease sets in abruptly; jaundice appears on first day.

Intensity: marked.

Fever: primary and secondary with intervening period of calm.

Associated characteristic features: black vomit, albuminuria, disproportion between pulse (which is slow) and the temperature.

Relapsing Fever

Onset: sudden, but jaundice occurs on second day.

Fever: lasts 7 days. It is then followed by an afebrile period of 7 days, and then fever returns to last 1 week.

Associated characteristic features: backache and muscular pain.

Icterus Neonatorum

Onset: jaundice appears on the second day after birth.

Intensity: both mild and severe forms are recognized.

Duration: the mild or the physiologic form lasts up to 2 weeks; the severe form proves rapidly fatal.

Syphilis of the Liver

Slight jaundice may occur in the **secondary stage,** and the liver may also be enlarged.

In **tertiary syphilis** of the liver we find:

onset: insidious.

intensity: slight.

liver: enlarged, nodular, very painful.

spleen: more often enlarged than in malignant disease.

gumma: also found elsewhere.

obstructive symptoms: present, as in atrophic cirrhosis. (q.v.)

Passive Congestion of the Liver

Jaundice is usually slight, but may become more marked if the catarrh extends into the bile ducts.
Liver: smooth, enlarged, pulsating, tender.
Lesions found in heart or lungs.
Usually runs a chronic course.

Malarial Fevers

Intermittent fever: jaundice rarely present except in severe cases.
Remittent fever: jaundice quite frequent.
Pernicious malaria: very severe hematogenous jaundice owing to the destruction of the red blood cells.

In conclusion it may be stated that jaundice is often associated with diseases of a purpuric nature and that jaundice is sometimes feigned by the use of drugs as rhubarb, santonin, saffron, turmeric, etc.

Concomitant Manifestations of Obstructive Jaundice

Icterus: skin, conjunctiva, and mucous membranes colored yellow.
Pruritus: an itching of the skin.
Bradycardia: a slow pulse in uncomplicated cases.
Hemorrhagic tendency: bleeding into skin and viscera may occur.
Fatty clay-colored stools: because no bile passes into the bowels.
Bile pigment in urine: color is dark brown like beer, and on standing a yellow foam occurs. Pigment is soluble in chloroform.
Localized sweating: palms or the abdomen.
Cholemia: signifies marked cerebral symptoms; as headache, irritability, and melancholia.

Comparison of Toxemic with Obstructive Jaundice

(Toxemic jaundice is due to the presence of poisons in the circulation which destroy the red blood cells.)

1. Jaundice is not as marked as in the obstructive varieties.
2. Clay-colored stools are absent.
3. Cerebral symptoms are more apt to be severe:
   vomit, delirium, and convulsions.
4. Usually runs a shorter period.
5. Pulse is rarely as slow as in obstructive jaundice.
6. Hemorrhages into the skin and mucous membranes occur in both forms of jaundice with equal frequency.

Passive Congestion of the Liver

Diagnosis:

1. Feeling of heaviness, tightness, and discomfort in the right hypochondrium, and tenderness on pressure over this area.
2. Dyspepsia, flatulence and tympanites.
3. It favors gastro-intestinal catarrh; usually diarrhea, but there may be constipation.
4. Interference in assimilation; therefore loss in weight, but this may not be noticeable, due to edema.
5. Slight degree of jaundice. It may be increased if the catarrh extends into the bile ducts.
6. Smooth, enlarged, and pulsating liver. If the liver is pressed upward manually the veins of the neck swell up.
7. The ascites in passive congestion is apt to come and go and persists for a long time, while ascites following cirrhosis of the liver bears a more serious prognosis as to the duration of the disease.
8. Chronic venous congestion predisposes to auto-intoxication. (Rolleston.)

The Cirrhoses of the Liver

The best classification of the cirrhoses of the liver is the **clinical classification,** viz.:

1. **Atrophic Cirrhosis of Laennec.** Gin Drinker's Liver, the Hob-nailed Liver, and Portal Cirrhosis.
2. **Hypertrophic Cirrhosis.**
3. **Syphilitic Cirrhosis.**
4. **Capsular Cirrhosis** or **Chronic Perihepatitis.**

Atrophic Cirrhosis

Definition: A chronic diffuse interstitial hepatitis characterized by an increase in connective tissue, but especially marked around the portal capillaries (interlobular connective tissue), which connective tissue tends to contract around these vessels, resulting in an obstruction of the blood flowing through the portal veins in the liver. The term "Portal Cirrhosis" is an excellent explanatory nomenclature for atrophic cirrhosis. Atrophy and contraction, however, occur late in the disease.

Note: however abundant this new connective tissue may be, we will fail to observe any of the so-called "obstructive symptoms" if there be proper compensation by the accessory circulation of the liver.

Symptoms: the disease is distinctly chronic in nature and the symptoms appear insidiously. The clinical symptoms are classified into (a) **obstructive** and (b) **toxemic.**

(a) **Obstructive symptoms;** due to stasis of portal veins:

1. **Chronic catarrh of the stomach,** giving rise to morning vomit, eructations, etc.
2. **Progressive ascites:** fluid shows characteristics of a transudate. If peritonitis develops, the fluid will show characteristics of an inflammatory exudate. Ascites is a bad prognostic sign.
3. **Splenic enlargement:** marked but obscured by the ascitic fluid. Best elicited by palpation.
4. **Gastro-intestinal hemorrhages:**
   Hematemesis is very prominent.
   Tarry (bloody) stools.
   Hemorrhoids (piles).
5. **Jaundice:** usually mild and occurs late.
6. **Œdema of the lower half of body** (due to ascites). (**Cardiac Hypertrophy** and emaciation very apt to develop and tubercular peritonitis frequently co-exists.)

(b) **Toxic symptoms:** at any stage of the disease there may occur: delirium, stupor or coma, and the condition may be mistaken for uremia.

The condition of the liver:

Early: uniformly and smoothly enlarged.
Second stage: rough, small, granular and contracted liver. There is no sharp transition from the first to the second stage. If ascites is marked, the fluid must be withdrawn in order to ascertain the size of the liver.

Hypertrophic Cirrhosis

Definition: a hyperplasia of the connective tissue of the liver showing a predilection for the connective tissue around the bile capillaries, which connective tissue on contracting interferes with the passage of the bile, causing intense jaundice; and also characterized by a permanent enlargement of the liver. The term "Biliary Cirrhosis" is very appropriate in contrast to Portal Cirrhosis.

Symptoms and signs:

Jaundice: occurs early and progresses. Icterus gravis with high fever and delirium may occur at any time. There are also the usual concomitant evidences of jaundice, but clay-colored stools may be absent.

Permanent smooth enlargement of the liver: the gall-bladder, however, is not enlarged.

Enlargement of the spleen: it is not, however, due to congestion, as in atrophic cirrhosis.

Recurrent attacks of pain in the region of the liver:

It is further distinguished from atrophic cirrhosis by the **absence of ascites** and other **obstructive symptoms,** by showing no tendency to occur in alcoholics, but shows a predilection chiefly for young persons.

Syphilitic Cirrhosis

Definition: This form of cirrhosis occurs in the tertiary stage of syphilis and is usually associated with gummatous formation in the liver.

Symptoms and **diagnosis:**

1. **Obstructive;** as in atrophic cirrhosis; i. e.: ascites, etc.
2. **Pain in region of liver;** but not always present.
3. **Jaundice** as a rule **not conspicuous.**
4. **Gumma found elsewhere.**
5. **Amyloid degeneration elsewhere.**
6. **Liver is enlarged. Nodular tumors** can be palpated. There are no umbilications of these nodules as we meet with in cancer of the liver.
7. **The X-ray** shows furrows between which, projections are outlined. These irregularities must be distinguished from cancer and cirrhosis.

Capsular Cirrhosis or Chronic Perihepatitis

Definition: A thickening of the fibrous capsule of the liver. There is no marked increase in the connective tissue in the substance of the liver proper. It may occur as a part of a widespread multiple serositis, which includes perisplenitis, proliferative peritonitis, adherent pericarditis, and mediastinitis.

Symptoms: are similar to those of atrophic cirrhosis excepting that there is no marked impairment of health (only near the end) and that toxic symptoms are always absent.

1. Jaundice: not common.
2. **Ascites:** always present and will recur after removal of the fluid. The ascitic fluid shows characteristics of an inflammatory exudate.
3. **Interstitial nephritis** apt to co-exist.
4. **Liver usually decreased in size.**
5. The disease **runs a protracted course,** even 15 years.

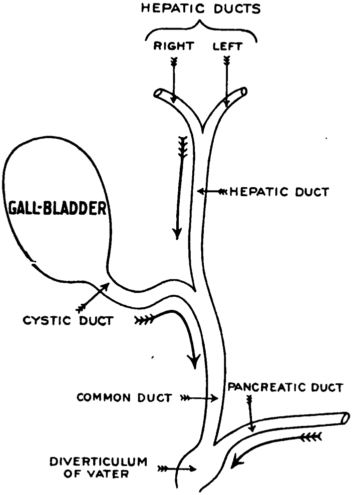
Cancer of the Liver

Diagnosis:

Age: 40 to 60 years.
Progressive emaciation, anemia, and cachexia.
Anorexia, nausea, and vomit.
Pain over liver; varies in intensity.
Mild degree of jaundice, but if common duct is blocked it becomes very intense.
Ascites is rare unless cancer is associated with cirrhosis. If such be the case, the liver is not enlarged.
Continuous fever: 100° to 102°.
Progressive, pronounced, irregular enlargement of the liver. The surface shows nodular areas with central depressions (Farrés Tubercles). Inasmuch as cancer of the liver is secondary to a cancerous growth in the portal system, we expect to find multiple tumors, both inside and outside the organ.
Spleen rarely enlarged.
Duration of the disease: not more than 1½ years.

Note: All hepatic tumors move with respiration, due to the inspiratory depression of the diaphragm pushing down the liver and all that is joined to it.

Gall-Stones



Diagrammatic representation of the Gall-Bladder and Ducts

Stones formed in the gall-bladder may remain in that organ without causing any appreciable symptoms or they may migrate through the ducts and when passing may be arrested at the cystic or the common ducts. The migration or ejection of the stone causes "biliary colic."

The accompanying features of a gall-stone colic are:

1. Sudden, agonizing paroxysmal **pains** in the upper region of the abdomen in the right hypochondrium. The pains radiate to the right shoulder.
2. Marked **chills;** and as a rule, moderate fever, 100° to 103°. Evidently some infection causes these chills.
3. Intense **sweating.**
4. **Vomiting.**
5. **Tenderness** in the region of the liver.
6. **Enlarged spleen** and sometimes a slight enlargement of the liver and gall-bladder.
7. Variable degree of **jaundice** (occurring only when stone is arrested in the common duct).
8. Gall-stone colic usually repeats itself at irregular intervals, and the attacks may vary in severity.

When the gall-stone is arrested in its passage through the CYSTIC DUCT, one of the following may occur:

1. Enlargement of the gall-bladder owing to an increase in its fluid contents. This condition is called "hydrops of the gall-

bladder." Such fluidity is an evidence of the chronicity of this condition. However, acute obstruction may also occur. The enlarged gall-bladder may be felt as a fluid tumor in the right hypochondrium projecting downward and perhaps a little toward the median line. In some cases the gall-bladder may subsequently atrophy. Jaundice, as a rule, is not conspicuous in obstruction of the cystic duct.

2. Suppurative cholecystitis or empyema of the gall-bladder may also occur. (See page 20.)

When the stone is arrested in the COMMON DUCT:

(A) **If the obstruction is complete and permanent:**

1. Jaundice is deep and lasting.
2. Septic features are absent.
3. Previous history of gall-stone colics.
4. Gall-bladder not enlarged.

(B) **If the obstruction is incomplete:** (Naunyn.)

1. Continuous or occasional presence of bile in feces.
2. Distinct variations in the intensity of jaundice and jaundice lasting more than one year.
3. Courvoisier's Law.
4. Normal or only slight enlargement of the liver.
5. Enlarged spleen.
6. Absence of ascites.
7. Fever.

The symptoms of incomplete obstruction further depend upon the presence or absence of an infective or of a suppurative cholangitis.

The term "Hepatic Intermittent Fever of Charcot" is given to certain cases of **"Ball Valve Stone"** in the Diverticulum of Vater, in which the following symptom-complex occurs:

1. Ague-like paroxysms of chill, fever and sweat.
2. Jaundice (variable in intensity) which deepens after each paroxysm, and persists for months and even years.
3. Pain in region of the liver and gastric disturbance accompanies the paroxysms.

If actual suppuration occurs in the bile ducts:

1. The fever is apt to be more remittent and without long intervals of apyrexia.
2. Jaundice is not so intense and does not deepen after paroxysms.
3. Liver is more apt to be enlarged, especially if it becomes the seat of an abscess.
4. Marked signs of septic poisoning.
5. Shorter duration.
6. Recovery seldom occurs.

DISEASES OF THE BILE DUCTS

(1) Catarrhal
(2) Suppurative

1. (a) Acute Catarrhal Angiocholitis

Definition: This represents a "mild" infection of the bile ducts (the infective agent not being definitely known). The synonym of this affection is "Catarrhal Jaundice," and is supposed to be due to a plugging of the terminal portion of the duct by inspissated mucus.

The most frequent history is as follows:

1. An attack of indigestion usually precedes.
2. Unexpected or sometimes gradual appearance of jaundice and its concomitant signs.
3. Young, comparatively healthy persons are usually the victims.
4. Jaundice rarely lasts longer than 6 to 8 weeks.
5. Fever is absent because the inflammation is mild.
6. There is little or no pain in the region of the liver.
7. Liver is only slightly enlarged, due to the retained bile.
8. The general health does not suffer.

1. (b) Chronic Catarrhal Angiocholitis

This results when there is a permanent obstruction of the common duct, no matter whether the obstruction be due to compression from without or something within the duct.

The **features** are:

1. Enlargement of the gall-bladder (most marked in complete obstruction).
2. Jaundice.
3. No fever except in incomplete obstruction, and then in the case of the latter, we may observe the "Hepatic Intermittent Fever."

2. (a) Suppurative Angiocholitis

Definition: This represents a **severe** purulent inflammation of all the bile ducts. This disease is the most serious sequel of cholelithiasis.

The usual history is as follows:

1. **Gall-stone colic** in most of the cases.
2. Signs of **septic poisoning,** due to the presence of pus; septic fever, leucocytosis, etc.
3. **Liver enlarged** and **tender.**
4. Variable degree of **jaundice.**
5. In a majority of cases, the pus drains into the gall-bladder, infects it, causes an infectious cholecystitis, and therefore an enlargement of the gall-bladder.
6. The ultimate results of septic poisoning are progressive emaciation and loss of strength.

2. (b) Acute Infectious Cholecystitis

Definition: This represents a severe inflammation of the gall-bladder, the causative agent being bacterial invasion. While gall-stones usually predispose to this disease, yet it may occur independent of gall-stones, especially in infectious fevers.

The usual history is as follows:

1. The patient has had attacks of gall-stone colics or is convalescing from an acute infectious disease, such as typhoid or pneumonia.
2. Sudden pain over the region of the liver. Local tenderness occurs later.
3. Nausea and vomit.
4. High fever and rapid pulse.
5. Prostration.
6. Jaundice: only in cases following gall-stones.
7. Gall-bladder enlarged upward and inward toward the foramen of Winslow.

The **dangers** are abscess, perforation, peritonitis, and the bowels may be implicated by adhesions and thus cause intestinal obstruction.

Abscess of the Liver

Frequent causes:

1. Ulcerations along the bowels causing suppurative pylephlebitis results in the so-called "Pyæmic Abscesses," which are usually multiple.
2. Dysentery causes the so-called "Tropical or Solitary Abscess."
3. Suppurative cholangitis due to gall-stones.

Rarer causes:

1. Extension from neighboring tissues. . . .
   - Subphrenic abscess.
   - Empyæma of the lung.
   - Empyæma of gall-bladder.
   - Pulmonary tuberculosis.
2. Trauma. . . . .
   - Contusion or rupture of liver.
   - Foreign body.
   - Penetrating wound.
3. Parasites. . . . .
   - Ascaris (thread worm).
   - Hydatid cyst (which may suppurate).
   - Dystoma Hæmotobium (in tropics).
   - Coccidia.
4. Infectious diseases.
5. Embolism through hepatic artery.

Symptoms and signs:

When abscess of the liver follows suppurative pylephlebitis (suppuration along the course of the portal vein), we will have in conjunction with the usual features of **sepsis** (chill, fever, and sweat), an **enlarged liver** and a slight degree of **jaundice.** The abscesses are multiple and the liver is smooth.

Leucocytosis is more apt to be present in pyæmic than in solitary abscess.

Owing to the numerous foci of infections these cases run a more acute course than solitary abscesses.

The **Solitary** or **Tropical abscess** is latent and runs a more chronic and protracted course. Its existence may be suspected when the following are present:

1. Intermittent (but irregular) septic fever, somewhat like malaria.
2. Pain, or rather a dull aching sensation, in the right hypochondrium. When peritoneum becomes involved, pains become sharp. Patient prefers to lie on right side. Pain and tenderness on pressure over liver region. If peritoneum is involved, a friction rub may be heard over the hepatic area.

3. Upward enlargement of the liver, the abscess USUALLY being in the right lobe of the liver. It may also be felt below the costal margin if the enlargement is great, and, if felt, the surface is smooth.
4. Slight jaundice, causing a muddy complexion.
5. A right purulent pleurisy is apt to occur as a result of the abscess perforating through the diaphragm.
6. Amœbæ may be discovered by aspiration.
7. The X-ray will detect abscesses of the posterior and superior portions of the liver. Abscesses projecting toward the abdomen show nothing by radioscopy. In the former case, abscesses of the convex surface cause the vault of the diaphragm to rise upward so that there is a difference of 5 cm. between the height of the dome on the right and that on the left. Another feature is the apparent immobility of the right dome of the diaphragm, which is painful, due to the contiguous inflammation. A tumor in that situation may also give similar signs when the X-ray is used. We must then be guided by the other signs and symptoms.

Acute Yellow Atrophy

Definition: An acute fatty degeneration of the liver resulting in a marked reduction in its size and by the finding of leucin and tyrosin in the urine. The heart and kidneys are usually concomittantly involved. The fact that a large spleen usually accompanies this disease is strongly suspicious of some infectious process, but the true cause is unknown.

Diagnosis:

1. **Sudden onset:** rarely do we have prodromata.
2. **Intense nervous manifestations:**
   - **1st stage:** is the **excitement stage,** in which occurs headache, sleeplessness, restlessness, progressive dulling of mentality, intense delirium (maniacal at times), and muscular twitchings.
   - **2nd stage:** is the stage of depression, which appears within 48 hours, and deep coma develops. Death occurs in the comatose stage and always within 4 days after the cerebral symptoms appeared.
3. **Early** and **progressive jaundice** and its associates.
4. **Marked splenic enlargement.**
5. **Pain** and **tenderness** may be elicited **in** the **hepatic area,** but is not as conspicuous as in phosphorus poisoning.
6. **Pulse:** early it is slow, later it is rapid. When pulse becomes rapid, severe vomiting appears.

7. **Cutaneous ecchymoses** and **internal hemorrhages** may occur in any part of the body.
8. The **reduction** in the **size** of the **liver** is most marked in the late stage.
9. **Fever:** normal or slightly elevated, but may rise greatly before death.
10. **Urinary findings:** bile pigment, leucin, tyrosin, reduction in urea while the amount of urine is almost normal.

Acute yellow atrophy must be differentiated from phosphorus poisoning. The characteristic features of the latter are:

1. **It sets in suddenly with vomiting** (the vomitus has a garlic-like odor), and with **severe epigastric pains.** A period of calm then ensues and lasts 48-72 hours.
2. Following the quiescent period, vomiting reappears, and with it, **diffuse abdominal pains,** most marked over the hepatic region.
3. The mind is clear except before death.
4. Jaundice appears late and is not very intense.
5. Spleen is seldom enlarged.
6. **Liver** is **enlarged** and **painful.**
7. Scanty urine.

(If sulphuric acid be added to the vomitus, and the mixture warmed, phosphorescent phenomenæ may be seen.)

Urinary Findings in Hepatic Disorders

1. Bile: in nearly all cases of jaundice. The color of the urine is therefore changed.
2. Urinary ammonia is increased in all hepatic disorders. (A large meat diet and all fevers do the same.)
3. The amino acids are, as a rule, increased in the cirrhoses.
4. If chronic constipation co-exists and causes auto-intoxication, then indicanuria is marked.
5. Leucin and tyrosin occurs in acute atrophy of the liver.
6. Urea is sometimes increased in hepatic congestion, but its determination in hepatic disorders will throw no light on the diagnosis. The same may be said of uric acid. It is said to be increased in disorders of the liver.
7. If a certain quantity of levulose be given to healthy individuals, alimentary levulosuria will occur in 10% of the cases. In hepatic disorders it will occur in 50%. The cirrhoses contribute the largest percentage of this figure. (Zeitschrift für Klinische Medizin, Vol. 72, Nos. 5 and 6.)

Specific Medication
in
Hepatic Disorders
with
Especial Reference
to
CHIONIA

The intimate association of the liver with the gastro-intestinal tract, by reason of the portal system of blood vessels and the action of bile during digestive processes, is a fact to be constantly borne in mind. In the so-called functional derangements of the liver we find such symptoms as are referable to the intestinal tract. It is therefore sometimes difficult to decide whether the primary cause is resident in the liver or in the bowels. However, we must acknowledge that poisonous substances formed in the intestines as a result of chronic constipation, auto-intoxication, etc., must eventually be carried to the liver by the portal blood vessels. Fortunately enough, nature has endowed the liver with the property of detoxifying these poisonous substances. But, when there is a too rapid accumulation of these poisonous substances, the liver will be unable to take care of this excess, and in some cases actual morphological changes will take place in the hepatic cells. Furthermore, the toxins, being absorbed into the system, react on the nervous system and give rise to the following nervous manifestations: headache, mental depression, dizziness, irritability, etc.

In order that the liver functionate properly we must regulate the bowels for the reason that constipation is a great cause of congestion (circulatory stasis) of the liver. For this purpose a mild laxative should be employed to deplete the circulation. In "Chionia" we find a remedy that will stimulate the portal circulation, hence removing the congestion of the liver. A slight laxative action can also be ascribed to this preparation, but it is rarely given alone for such purposes. When a thorough evacuation is desired it is best to use sodium phosphate in conjunction with Chionia. Under the influence of Chionia the liver and intestines are encouraged to resume their normal functionation.

That condition commonly spoken of as "*biliousness*" can be ascribed to the torpidity of the liver, which, in turn, causes a torpidity of the digestive functions. Hence we find an improperly functionating liver and the result is smaller quantity of bile is poured into the intestines. Physiology teaches us that the amount of bile secreted is directly dependent upon the quantity and composition of the blood flowing through the liver, hence, whenever portal stasis occurs the quantity of bile is decreased. One fact which becomes evident in such cases is the change in the character of the stools. Bile is credited with a specific action on fats and a slight antiseptic action in the intestines. Inasmuch as the bile (the quantity being reduced) does not act completely on all the fat ingested, the stools are found to be yellowish-gray, and in other cases of complete biliary retention, they are clay-colored. While the antiseptic action of bile is only slightly marked, still its presence in the bowels in normal quantities serves to inhibit intestinal putrefaction and toxin formation with its ultimate nervous symptoms, as before explained.

The fact that bile itself stimulates intestinal peristalsis must not be forgotten in the treatment of our patients. Naturally with a reduction in the quantity of bile secreted, we will expect to see the bowels become sluggish in proportion to the decrease. In the treatment we must aim to increase the flow of bile, and such drugs which accomplish this purpose are called "cholagogues" or "bile persuaders." While podophyllin and mercurials are employed as such in cases of hepatic torpor, these cholagogues are somewhat drastic in their action, and, furthermore, if calomel be employed carelessly and for a long period, we must not overlook the danger of mercurial poisoning. An efficient cholagogue possessing a mild laxative action is to be had in "Chionia." A point in favor of this medication is not only the absence of severe purgation but that it stimulates the secretion of bile long before its laxative action is noticeable.

The restoration of the proper biliary secretion results in the reappearance of the normal processes of digestion, absorption, and assimilation of food, toxins are prevented from accumulating in the bowel and acting on the nervous system, the stools assume their natural color, and the bowels become regular. Chionia acts specifically as a stimulant to the biliary ducts, causing a contraction of their muscular coats, and it also depletes a congested liver.

Being familiar with the manifold causes of **jaundice,** it would be natural to ask: Are all cases to be treated alike, regardless of the cause, and whether or not we have a specific for this symptom? It would be folly to try to dislodge a malignant tumor implicating the bile ducts so as to secure the patency of the bile ducts with the same remedy as one would employ in catarrhal jaundice to remove the catarrhal exudate and thus open and drain the bile ducts. In the first case surgical intervention is the only hope, while in the latter, we can state a more favorable prognosis. The judicious administration of MILD laxatives, alkaline mineral waters, and proper dieting, will in 6 to 8 weeks result in disappearance of all discolorations of the skin. Jaundice must not be treated *per se,* as it is only a symptom. The pathological condition responsible for this pigmentation must be discovered, and our attack directed there. We must remember the hematogenous causes of jaundice, e. g., malaria, in which case we attack the parasites in the blood by **quinine** which is a specific in malaria and by killing the parasites we prevent further destruction of the red corpuscles. Those cases of jaundice which owe their origin to passive congestion of the liver secondary to cardiac and pulmonary disease will improve greatly by toning the heart with suitable tonics and proper depletion of the liver by gentle laxatives. Nature oftentimes accompanies a thorough depletion by hematemesis or by a hemorrhage from the bowel. Chionia is very well adapted in the treatment of hepatic congestion owing to its specific action in depleting the portal circulation. Toxemic jaundice will be found very persistent to treat.

The **iodides** occupy an undisputed place in the book of treatment of hepatic disorders, more especially in the treatment of syphilis of the liver. The iodides are specific and must be pushed in large doses. The iodides have also been used in catarrhal jaundice and in passive congestion and have given good results. (It is claimed that by reducing the viscosity of the blood they inhibit the tendency to stasis of the blood, stasis being a predisposing cause of catarrh of the ducts.)

Ipecac is a remedy that will afford relief in a case of acute hepatitis arising from an attack of dysentery. However, when abscess formation occurs in the liver little can be accomplished by using this drug. In the case of multiple abscess we have to deal with a systemic intoxication and the patient must be treated for such. The only case of abscess that is amenable to operation is the solitary abscess, and even then it should be left alone if the patient is expectorating pus.

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